

TABLE 26. STATISTICAL RESULTS OF ANIMAL HEALTH PROFILE

Parameter	Set 1		Set 2		Set 3	
	T	R	T	R	TR	R
Glucose	<0.0001		0.0030			
BUN	0.0366	<0.0001	0.0001	<0.0001	0.0394	0.0024
Creatinine	0.0037		0.0004			
Na+	0.0060		0.0002			
K+			0.0138			
Cl-				0.0146		
CO ₂			<0.0001			
Uric Acid	0.0004		0.0122			
Ion Gap	0.0132		<0.0001			
Ionized Ca		0.0108	<0.0001	0.0284	0.0019	0.0111
Calcium		0.0187	<0.0001		0.0125	
P	<0.0001		<0.0001			
Alka. Phos.	0.0115	0.0003	0.0156	0.0025		0.0276
LDH			0.0405			
SGOT	0.0054	0.0684			0.0287	
SGPT	0.0234					
Cholesterol		0.0032	<0.0001			0.0394
Triglycerides			0.0007			
Total Protein						
Albumin	0.0016		0.0056			
Globulin						
A/G	0.0020					
BUN/Cre	<0.0001	<0.0001	<0.0001	0.0128		
T ₄	0.0001	0.0235	<0.0001			
Albumin	0.0007		0.0008			
Alpha 1						
Alpha 2	<0.0001		0.0003			
Beta				0.0168		
Gamma	0.0010					
A/G	0.0002		0.0023			
WBC						
RBC						
HGB			0.0431			
HCT						
MCV		0.0009				
MCH		0.0472				
MCHC			0.0173			
Lymphocyte	0.0065		0.0412			
Neutrophil	0.0055					
Mono						
Eosin						

T: Temperature effect

R: Radiation effect

TR: Temperature - radiation effect

The means and standard errors of the corticosterone level in rats exposed to various incident power densities and environmental temperatures are shown in Figure 25. Analysis of variance of the data at each temperature showed significant difference only at 17.8°C ($f = 3.43$, $df = 3,35$, $p < 0.05$). For the sham and 5-mW/cm² groups, two-way analysis of variance detected no effect of temperature or exposure.

At low temperature (17.8°C), the corticosterone level was low (2.1 µg/100 ml) but increased while exposed to microwaves, especially at 15 mW/cm². The difference was statistically significant. At 22.2°C, however, the level decreased when compared to control, although not statistically significant. Animals died during the 15-mW/cm² exposure at medium and high temperatures; obviously this was highly stressful. Then why did the corticosterone level not show this effect since it is related to stress? Possibly the observed effect was due to adrenal exhaustion when exposed to chronic stress (Selye, 1950). This is often seen in aging rats, when the pituitary-adrenal-system functioning remains intact but the reserve capacity to respond to stress is diminished (Hess and Riegler, 1970).

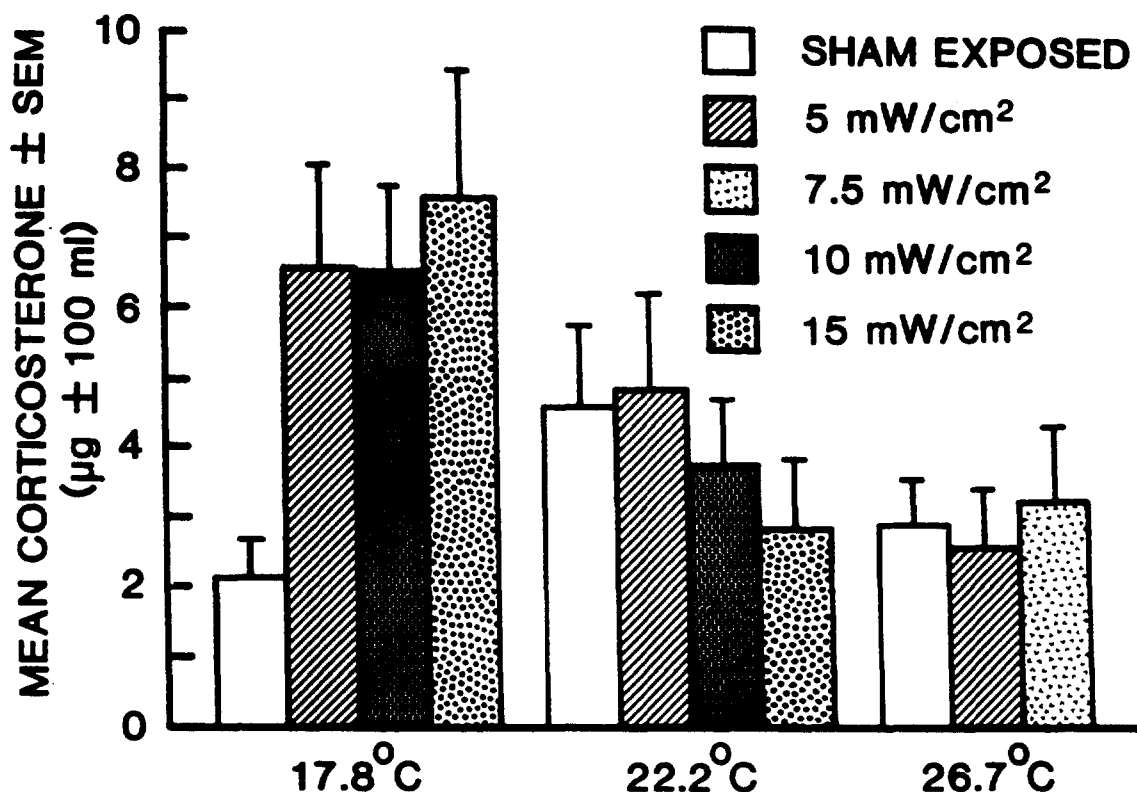


Figure 25. Corticosterone levels in rats exposed to various levels of microwave radiation under three environmental temperatures.

Oxygen Consumption and Carbon Dioxide Production

Table 27 lists the O_2 consumption and CO_2 production of the rats exposed to 5, 7.5, 10, or 15 mW/cm^2 at 17.8, 22.2, or 26.7°C. No data for sham-exposed rats are shown. When the sham-exposed rat was in the metabolism cage, the empty cage provided no data that could be used for the correction of the drift in equipment. However, when the sham and exposed animals were measured simultaneously, the differences between them were obtained, as shown in Table 28. An example of daily variations of the data is shown in Figure 26. The data show a consistent decrease of both O_2 and CO_2 production for all exposures.

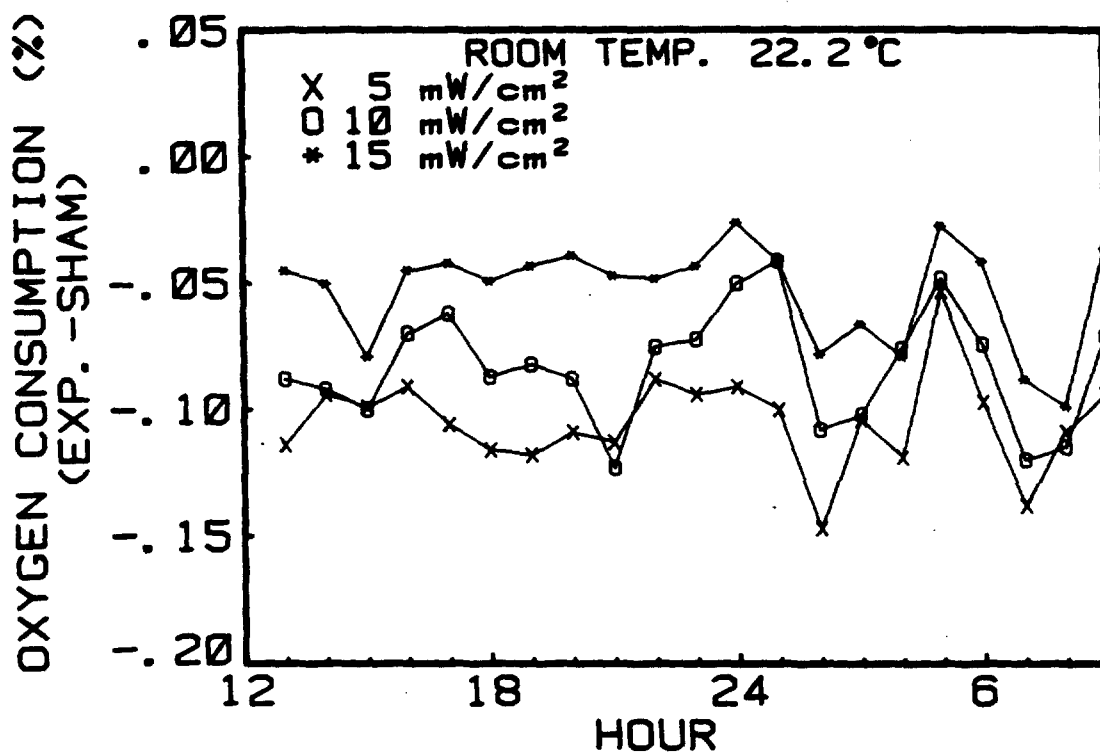


Figure 26. Example of daily oxygen-consumption variation (averaged over 6 wk) at 22.2°C.

TABLE 27. OXYGEN CONSUMPTION (%) AND CARBON DIOXIDE PRODUCTION (%) OF RATS EXPOSED TO MICROWAVE RADIATION UNDER THREE ENVIRONMENTAL TEMPERATURES

Exposure Time (mW/cm ²)		17.8°C		22.2°C		26.7°C	
		Mean	SD	Mean	SD	Mean	SD
<u>O₂ Consumption</u>							
5	e l	0.325	0.022	0.348	0.011	0.369	0.047
		0.307	0.021	0.388	0.022	0.323	0.083
7.5	e l					0.382	0.043
						0.305	0.128
10	e l	0.308	0.055	0.350	0.017		
		0.320	0.016	0.397	0.023		
15	e l	0.278	0.053	0.367	0.072		
		0.277	0.031	0.427	0.039		
<u>CO₂ Production</u>							
5	e l	0.205	0.065	0.241	0.026	0.160	0.013
		0.223	0.013	0.247	0.022	0.153	0.022
7.5	e l					0.164	0.015
						0.130	0.057
10	e l	0.172	0.056	0.213	0.023		
		0.192	0.007	0.224	0.028		
15	e l	0.173	0.055	0.217	0.032		
		0.196	0.015	0.247	0.022		

e and l = early and late recordings for 6-wk experiment

TABLE 28. CHANGE OF OXYGEN CONSUMPTION (%) AND CARBON DIOXIDE PRODUCTION (%) OF RATS EXPOSED TO MICROWAVE RADIATION, RELATIVE TO SHAM-EXPOSED RATS, UNDER THREE ENVIRONMENTAL TEMPERATURES

Exposure (mW/cm ²)	Time	17.8°C		22.2°C		26.7°C	
		Mean	SD	Mean	SD	Mean	SD
<u>O₂ Consumption</u>							
5	e	-0.068	0.080	-0.122	0.032	-0.053	0.053
	l	-0.068	0.020	-0.101	0.005	-0.037	0.013
7.5	e					-0.075	0.040
	l					-0.030	0.022
10	e	-0.093	0.031	-0.101	0.011		
	l	-0.115	0.022	-0.042	0.011		
15	e	-0.105	0.079	-0.090	0.004		
	l	-0.122	0.014	-0.045	0.010		
<u>CO₂ Production</u>							
5	e	-0.030	0.032	-0.091	0.013	-0.043	0.019
	l	-0.050	0.030	-0.065	0.001	-0.047	0.016
7.5	e					-0.054	0.020
	l					-0.053	0.001
10	e	-0.072	0.043	-0.113	0.001		
		-0.099	0.011	-0.083	0.017		
15	e	-0.059	0.037	-0.108	0.005		
	l	-0.075	0.014	-0.078	0.019		

e and l = early and late recordings for 6-wk experiment

Statistical analysis of the O_2 data was done by sets as before, except there was no sham-exposed group here. In rats exposed to 5, 10, or 15 mW/cm^2 at 17.8 and 22.2°C, the effects of temperature ($f = 98.9$, $df = 1,36$, $p < 0.0001$); temperature-radiation ($f = 7.44$, $df = 2,36$, $p = 0.002$); time ($f = 7.96$, $df = 1,36$, $p = 0.0077$); and time-temperature ($f = 9.54$, $df = 1,36$, $p = 0.039$) were significant. Summaries of least significant differences are shown in Table 29. Differences in O_2 consumption were not significant between underscored conditions; differences not underlined were statistically significant at $p < 0.01$. When analyzed separately for 17.8 and 22.2°C, the effect of radiation was significant for 17.8 ($f = 5.22$, $df = 2,18$, $p = 0.0163$) but not for 22.2°C. At 22.2°C there was a significant effect of time ($f = 17.56$, $df = 1,18$, $p = 0.0006$).

TABLE 29. COMPARISON OF OXYGEN CONSUMPTION IN RATS EXPOSED TO 5, 10, OR 15 mW/cm^2 AT 17.8°C (L) OR 22.2°C (M)

a) Averaged across time

L - 15 L - 10 L - 5 M - 5 M - 10 M - 15

b) Averaged across radiation

l - L e - L e - M l - M

Magnitudes increase from left to right
e, l = early and late recordings

At 5- mW/cm^2 exposure, the effect of temperature alone was not significant, but it was significant for time-temperature interaction ($f = 9.83$, $df = 2,18$, $p = 0.0013$). The groups are (l-L, l-H, e-L, e-M, e-H) and (e-L, e-M, e-H, l-M). Radiation had no effect on O_2 consumption at 26.7°C (H), but early consumption was significantly higher than late consumption ($f = 6.23$, $df = 1,11$, $p = 0.0297$).

Carbon dioxide production was analyzed in the same way. In rats exposed to three power densities at 17.8 and 22.2°C, effects of temperature ($f = 23.65$, $df = 1,36$, $p < 0.0001$); radiation ($f = 4.71$, $df = 2.36$, $p = 0.0151$); and time ($f = 5.91$, $df = 1,36$, $p = 0.0202$) were significant. When compressed over time, the data can be grouped into "17.8°C: 10, 15, 5 mW/cm²" and "17.8°C: 5 mW/cm²; 22.2°C: 10, 15, 5 mW/cm²." When analyzed separately for 17.8 and 22.2°C, the effect of radiation was not significant for either temperature. The time effect at 22.2°C showed higher CO₂ production late in the exposure rather than early. The effect of temperature at 5-mW/cm² exposure was highly significant ($f = 25.77$, $df = 2,18$, $p < 0.0001$). Data can be grouped into "26.7°C" and "17.8°C, 22.2°C." At 26.7°C, radiation had no effect on CO₂ production.

Statistical Methods. Rats available for study at the end of each of the three 6-wk temperature experiments were killed and assayed according to the following plan:

Units of variation were all based on one observation per animal. For the flow cytometric variables, each observation consisted of a single measurement from one 20,000-cell sample analyzed. For the in vitro function tests of lymphocyte activation, each observation was the average of three determinations for an individual rat. For the CFU-C assay, each observation was an average of two replicate samples.

Data for each variable were considered in two analysis-of-variance (ANOVA) contexts. Sham and exposed rats (0 and 5, 10, and 15 mW/cm² respectively) observed at 17.8°C and 22.2°C were included in a general mixed-model design that was analyzed via maximum-likelihood estimation (MLE) under the BMDP3V statistical computer program (BMDP Statistical Software, 1983). Radiation dosage and temperature served as completely crossed fixed factors with four and two levels respectively. Day of sacrifice served as a random factor with six levels (days); three levels nested in each temperature level and completely crossed with the four radiation levels. Control (sham) animals were pooled across alcoves with sacrifice-day membership preserved to achieve a reasonably balanced design. However, complete or proportional balance was still unobtainable, thus necessitating the MLE approach to the mixed model. The model saturated in the fixed effects (RFR, temperature, RFR-temperature interaction) and all possible subset models controlling for day of sacrifice were evaluated via change in deviance under the Chi-square likelihood ratio test and subsequent consideration of MLE/standard-error ratio for individual effects. In a similar manner significance of the random effect associated with day of sacrifice was evaluated against the fixed-effects saturated model. In order to identify outlier-dependent effects, all models were evaluated with and without extreme outliers.

Sham and exposed (5 and 7.5 mW/cm²) rats observed at 26.7°C were included in a proportionally balanced mixed-model design that was analyzed via standard ANOVA methodology. Radiation dosage served as a fixed factor with three levels. Day of sacrifice served as a random factor with two levels which were completely crossed with the three radiation levels. Sham animals were again pooled across alcoves with sacrifice-day membership preserved. In this instance proportional balance in cell size was achieved, insuring orthogonality of sums of squares. This allowed evaluation of overall F tests for interaction and main effects of the fixed and random factors. Data sets with extreme outliers were reanalyzed in a Winsorized fashion in order to identify outlier-dependent effects.

Results

The statistical model used in this study indicated that statistically significant temperature and radiation effects were associated with many of the variables considered. Close examination of respective MLEs and their standard errors showed that only some of the effects were of biological significance; the raw data is presented. Table 30 shows the effects of radiation and temperature on cellularity of the spleen and thymus. With the exception of the spleen at 0 mW and 17.8°C, at any one exposure level, cellularity with these organs tended to decrease as temperature increased. However, increasing radiation dose from 0 to 5 and then 5 to 10 mW/cm² tended to increase cellularity. At the 15-mW/cm² dosage, the number of cells decreased to below numbers for the unexposed group. For the spleen the radiation effect was significant ($p < 0.05$), and for the thymus the radiation and temperature effects were significant ($p < 0.05$ each).

The following is a list of statistical-effects ($p < 0.05$) abbreviations used in the tables.

Outlier dependent

Radiation	R	r
Temperature	T	t
Day	D	d
Interactions	RT, RD	rt, rd
No effect	-	
Not calculated	X	

TABLE 30. CELLULARITY OF THE SPLEEN AND THYMUS IN ANIMALS UNEXPOSED AND EXPOSED TO RADIOFREQUENCY RADIATION

Exposure level (mW/cm ²)	Cells counts per organ x 10 ⁶ [mean(SD)]			
	Spleen ^a		Thymus ^b	
	17.8°C	22.2°C	17.8°C	22.2°C
0	211(44)	226(62)	232(126)	145(64)
5	225(42)	198(67)	296(72)	180(75)
10	213(69)	204(64)	250(74)	150(95)
15	158(60)	131(25)	181(50)	132(66)

a: R

b: R, T

The response of hematopoietic progenitor cells to radiation and temperature conditions is depicted in Table 31. Radiation and temperature had significant effects on the numbers of macrophage and total colonies generated. For example, the number of macrophage colonies rose from 78 and 38 at 0 dosage to 105 and 46 at 10 mW/cm², respectively, at 17.8 and 22.2°C; at 15 mW/cm² the responses were equal to or lower than control values. This same pattern of responsiveness was observed for the granulocyte, mixed, and total-colony responses. The temperature effects within a given exposure group reveal that macrophage, granulocyte and total-colony responses each decreased approximately 50%; the shams also responded in this manner. Statistical analysis revealed an interactive effect between temperature and radiation in the response of mixed-colony progenitors. The exact biological significance of this is unclear.

TABLE 31. RESPONSE OF HEMATOPOIETIC PROGENITOR CELLS TO VARYING TEMPERATURES AND RFR DOSES

Exposure level (mW/cm ²)	Number of CFU per 10 ⁵ Marrow Cells Plated(SD)			
	17.8 ⁰ C	22.2 ⁰ C	17.8 ⁰ C	22.2 ⁰ C
	Macrophage		Granulocyte	
	0	78(19) ^a 38(7)	19(8) ^b 6(6)	
	5	93(16) 43(18)	30(13) 9(6)	
	10	105(25) 46(9)	24(7) 9(2)	
	15	77(20) 32(12)	30(14) 7(3)	
	Mixed		Total	
	0	14(7) ^c 18(8)	111(20) ^d 61(12)	
	5	20(8) 16(6)	143(35) 68(27)	
	10	14(3) 31(7)	143(25) 86(13)	
	15	19(5) 20(10)	125(37) 58(17)	

a = rt, R, T, D

b = r, T, d

c = RT, R, T, D

d = R, T, D.

The functional activity of T- and B-cell lymphocyte populations from the spleen and thymus was examined by stimulation in vitro with various mitogens such as LPS, Con A, PWM, PHA, and PPD. Although statistically significant effects (RT, R, and T) were observed in many of the mitogen variables, close examination of respective MLEs and their standard errors revealed no biologically meaningful differences in any of the "statistical cells" analyzed. Table 32 summarizes the statistical effects that were found. Immunofluorescence flow cytometric analysis of the populations of cells in the bone marrow, spleen, and thymus gland, using antisera-defining cell-surface differentiation antigens present on B cells (s-Ig) and T cells (Thy 1.1), is summarized in Table 33.

TABLE 32. SUMMARY OF STATISTICAL EFFECTS FOUND BY VARYING THE TEMPERATURE AND DOSE OF RFR ON THE IN VITRO MITOGEN RESPONSIVENESS OF SPLENOCYTES AND THYMOCYTES

Spleen	17.8-22.2°C	26.7°C	Thymus	17.8-22.2°C	26.7°C
<hr/>					
Observed scale					
<hr/>					
LPS	-	RD	Con A	D	R
Con A	RT, D	-	PWM	D	r
PWM	T	D	PHA	T, d	rd, R
PHA	R	RD			
PPD	T	D			
Log Scale					
<hr/>					
LPS	-	RD, r	Con A	D	D
Con A	RT, D	RD, R	PWM	T, D	R
PWM	T	RD, R	PHA	T, D	RD
PHA	R	RD			
PPD	t	D			

TABLE 33. SUMMARY OF STATISTICAL EFFECTS FOR THE FLOW CYTOMETRIC DATA
COMPARING MARROW, THYMUS, AND SPLENOCYTE B- AND T-CELL
POPULATIONS UNDER VARYING CONDITIONS OF TEMPERATURE AND RFR

		Significant Effects Observed					
		Bone Marrow		Spleen		Thymus	
Stain	Population analyzed	17.8- 22.2°C	26.7°C	17.8- 22.2°C	26.7°C	17.8- 22.2°C	26.7°C
B cell							
	Viable cells						
	Peak intensity	D	D	-	D	D	X
	Mean intensity	D	D	D	D	D	RD
	% lymphocytes	T, D	D	R, D	D	D	R, D
	Peak lymphocytes	r, T, D	D	D	D	D	X
	Mean lymphocytes	R, D	D	D	D	D	D
	% viable cell with s-Ig	T, d	R, D	D	-	RT, D	R
	% lymphocyte with s-Ig	R, D	RD,	D	D	RT, D	-
T cell							
	Viable cells						
	Peak intensity	T	D	T, D		d	R
	Mean intensity	T, D	D	R, T, D	-	D	R, D
	% lymphocytes	T, D	-	T, D	-	rt, r T, D	D
	Peak lymphocytes	R, D	D	D	D	d	D
	Mean lymphocytes	T, D	R, D	RT, R, T, D	D	D	R, D
	% viable cell with Thy 1.1	D	R, D	T, D	D	r, D	-
	% lymphocyte with Thy 1.1	D	R, D	D	D	rt, r, T, d	-

Only a summary form of the statistical analysis is presented since few biologically meaningful differences were observed. Those differences deemed important are shown in Tables 34 and 35. In Table 34 a pronounced temperature effect can be observed when the percent of B cells expressed as a fraction of all viable marrow cells is considered as a function of temperature and radiation exposure levels. General trends included an increasing fraction of B cells at 22°C and a substantial decrease at 26.7°C. A radiation effect was noted only at 26.7°C. In Table 35 a clear-cut radiation effect is evident when the fraction of B cells of all viable cells is examined as a function of radiation dose. From 0 to 7.5 mW/cm², the fraction of B cells increased from 2% to 5% of all cells.

TABLE 34. FRACTION OF BONE MARROW CELLS DETECTED BY FLOW CYTOMETRIC TECHNIQUES USING ANTIBODIES SPECIFIC FOR B CELLS

Exposure level (mW/cm ²)	% B cells (SD)		
	17.8°C ^a	22.2°C ^a	26.7°C ^b
0	12(5)	13(2)	8(2)
5	10(2)	12(2)	10(3)
7.5	c	c	7(2)
10	10(2)	14(3)	c
15	11(1)	14(3)	c

a = T

b = R

c = not tested

TABLE 35. FRACTION OF THYMOCYTES DETECTED BY FLOW CYTOMETRIC TECHNIQUES USING ANTIBODIES SPECIFIC FOR B CELLS

Exposure Level (mW/cm ² /26.7°C)	% B cells(SD)
0	2(2)
5	3(3)
7.5	5(3)

R, p = 0.055

This study was designed to evaluate the immunologic status of rats exposed to three environmental temperatures (17.8, 22.2, and 26.7°C) and to 0, 5-, 10-, and 15-mW/cm² RFR over a 6-wk period. Alterations in the hematopoietic and immunologic networks have been reported in animals exposed to RFR at and below 10 mW/cm², but such effects could be attributable to thermal changes within the animals. The experiments we report here demonstrate alterations in the hematopoietic and immunologic systems of rats exposed to various temperatures and levels of RFR. Dramatic effects observed were as follows: (a) A pronounced radiation effect ($p < 0.05$) on the cellularity of the spleen where a 30% decrease in the numbers of recoverable viable cells occurred after 15-mW/cm² exposure; elevating the temperature from 17.8 to 22.2°C did not produce any additional changes. (b) A dramatic radiation and temperature effect for the cellularity of the thymus gland in which a biphasic response was noted. Exposure to 5 mW/cm² at both 17.8 and 22.2°C produced a 20 to 30% increase in numbers of recoverable viable thymocytes compared to the unexposed animals; however, the elevation in temperature alone produced nearly 30% reductions in cellularity. Stimulatory and inhibitory effects of RFR on the immune response have been reported previously although not under these experimental conditions. Bowhill (1981) and Smialowicz (1982a) suggest that immunological competence tends to change in rats exposed to microwave radiation at any SAR in excess of their basal metabolic rate. Bowhill has postulated a biphasic response of the immunological apparatus after RFR exposure. Our data is consistent with such a hypothesis. The basis for this response could best be explained by thermal stress and the ensuing process of thermoregulation.

The response of the hematopoietic progenitor cells of the marrow to these conditions was very different from that after 6 and 12 mo of exposure to low levels of RFR (480 μ W/cm²). The numbers of total colonies and macrophage, mixed, and granulocyte colonies from animals exposed at 17.8°C demonstrated a significant radiation effect ($p < 0.05$) at low RFR levels (5 and 10 mW/cm²) where the number of colonies increased (20-70%)

but then decreased somewhat. The response at 22.2°C was decreased by 2- to 3-fold at every exposure level, with the exception of the mixed-colony response which appeared insensitive to the effects of elevated temperature. Comparing these progenitor cell responses to those of animals exposed to low levels of RFR for 6 and 12 mo reveals differences. Although colony types were reduced in numbers and were distributed differently, in this study a 10-fold increase in the RFR dose generally produced a 30-70% increase in colonies. This effect was temperature dependent but not observed consistently at 22.2°C. The biological basis for these findings is unclear and awaits further investigation.

Among the many variables examined by immunofluorescence cytometry, only two appeared to have significant biological implications. The first is a decrease in the percentage of B cells in the marrow, which was independent of radiation but highly dependent on temperature. With no RFR a temperature increase of 4.4°C produced a 30% decrease in marrow B cells. No such effect was seen in exposed animals. The frequency of B cells in the thymus gland is generally low (< 2.0%); the levels of thymic B cells in sham-exposed animals were consistent with this observation. Exposure to 7.5 mW/cm² of RFR, however, doubled the levels of thymic B cells. In the previous 6- and 12-mo study conducted at the University of Washington, the composition of the lymphoid cells within the marrow underwent only relatively minor alterations--RFR produced a marginally significant (p = 0.05) reduction in the fraction of marrow B cells. Our observation of increased levels of B cells within the thymus gland is in marked contrast to that of the previous study after 12-mo exposure. That microwave radiation produces alterations in the migration of lymphoid cells has been reported. Such an explanation might apply to our findings of increased B cells in the thymus. Several interpretations exist, but the one most plausible is that radiation increases B-cell migration to the thymus from the blood. Further experiments would be required to substantiate this notion.

The statistical approach used in this study requires some comment. The general mixed-model design for the experiments at 17.8°C and 22.2°C was analyzed via maximum-likelihood estimation. This method was necessary to

account for random-day effects against the fixed effects of RFR, temperature, and RFR-temperature interaction in an unbalanced design. Due to the small number of animals used in this study, the biological significance of variable effects with $p < 0.05$ was posited by examining the magnitude and variability of the MLEs for these effects. For many of these variables no body of literature exists that defines normal values and the expected variations within a normal rat population. Some variables measured are intrinsically precise and accurate; for example, the flow cytometric variables generally show small variations ($< 10\%$) about means. By contrast, measurements of mitogen stimulation of cells in vitro usually show much greater variation (30-80%) about the means. The magnitude of responses considered to be biologically meaningful can be seen in the AIDS literature where absence or reduction of response by 80% are deemed biologically significant.

Gross Pathological and Histopathological Evaluation

Tables 36-38 list the lesions of the animals exposed to 0, 5, 7.5, 10, and 15 mW/cm^2 at 17.8, 22.2, and 26.7°C environment. Necropsy of the animals that died during the exposure showed pulmonary hemorrhage and edema apparently due to the heat stress. For all three temperatures, high lesions occurred in the lungs. Since each temperature study lasted only 6 wk, not many lesions were found. Statistical analysis would not be meaningful because of not only the sparse data but also the uneven sample size and exposure time due to early death during high-power exposure.

TABLE 36. HISTOPATHOLOGICAL RESULTS OF 6-WEEK EXPOSURE*
AT 17.8°C

Lesions	Power Density (mW/cm ²)			
	0	5	10	15
Adrenal				
foci of cortical cellular alteration	1			
Aorta				
multifocal and mineralization				1
Epididymus				
sperm granuloma			2	
Kidney				
membranous glomerulonephritis				1
cystic collecting tubule	4	2	2	2
Liver				
acute hepatic congestion				1
Lung				
acute pulmonary congestion and edema				1
acute petechial agonal pulmonary hemorrhages				1
peribronchiolar lymphoreticular cell proliferation	6	7	7	7
Mandibular sg				
nonsuppurative periductal sialoadenitis	1	1	1	
Pancreas				
pancreatic ductal lithiasis			1	1
Stomach				
diffuse gastric hyperkeratosis			1	
gastric hyperkeratosis		1		1
Thymus				
multiple echymotic hemorrhages				1

*Ten animals in each group.

TABLE 37. HISTOPATHOLOGICAL RESULTS OF 6-WEEK EXPOSURE*
AT 22.2°C

Lesions	Power Density (mW/cm ²)			
	0	5	10	15
Adrenal				
focal adrenal cortical ossification			1	
foci of cellular alteration	2			1
Brain				
pituitary cyst				1
Heart				
cardiomyopathy		1		
Kidney				
cystic tubules	2		1	1
Liver				
nonsuppurative hepatitis	1			
Lung				
peribronchiolar lymphoid cell proliferation	9	7	7	5
Pancreas				
cystic pancreatic ductal lithiasis				1
Pituitary				
cyst	1			
Salivary gland				
nonsuppurative periductal adenitis	1		1	

*Ten animals in each group.

TABLE 38. HISTOPATHOLOGICAL RESULTS OF 6-WEEK EXPOSURE*
AT 26.7°C

Lesions	Power Density (mW/cm ²)			
	0	5	7.5	10
Adrenal				
foci of cellular alteration	1			
Heart				
focal cardiomyopathy	1	1		
focal cardiomyopathy				1
focal ectopic myocardial ossification			1	
Kidney				
hydronephrosis	1			
renal collecting tubules	1			
renal cortical cyst		1		
multiple cystic collecting tubules		1		
Liver				
peribronchiolar lymphoreticular cell proliferation			1	
Lung				
acute pulmonary congestion				1
peribronchiolar lymphoreticular cell proliferation	9	8	5	
Pancreas				
pancreatic ductal lithiasis	1			
Preputual gland				
cystic preputual gland hyperplasia	1			
Stomach				
gastric hyperkeratosis	1			
Urethra				
proteinaceous urethral calculi				1
Zymbals gland				
nonsuppurative periductal adenitis			1	

*Number of animals in each group: 0 (10)
5 (10)
7.5 (8)
10 (1)

CONCLUSIONS

Effects observed in the original long-term exposure study--the increased corticosterone at 6 wk, the increased B and T cells after 13 mo, and the effect on mitogen stimulation--were not replicated in this study. Instead, the only consistent effect that we observed in both 6- and 12-mo exposed rats was the increase in hematopoietic progenitor cells in their bone marrow. Confirmation and further studies of this effect are needed.

In this study when both the radiation and environmental temperature were raised to high levels, the effects seen were mainly on thermoregulation which is a complex function of power level and environmental temperature.

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